THE ANATOMICAL RECORD PART A 281A:1088–1094 (2004)

Three-Dimensional Structure and Evolution of Primate Primary Visual Cortex

ELIOT C. BUSH* AND JOHN M. ALLMAN
Biology Division, California Institute of Technology, Pasadena, California

ABSTRACT

In this study, three-dimensional reconstructions of primate primary visual cortex (V1) were used to address questions about its evolution. The three-dimensional shape of V1 in anthropoids is significantly longer and narrower than in strepsirrhines. This difference is an effect of clade and is not due to differences in activity pattern or V1 size. New measurements of V1 volume were also provided in order to reassess V1 size differences between strepsirrhines and anthropoids. It was found that for a given lateral geniculate nucleus (LGN) volume, anthropoids have a significantly larger V1 than strepsirrhines do. This is important since LGN is the principal source of V1's input. Finally, independent contrasts analysis was used to examine the scaling of V1 relative to LGN, the rest of cortex, and the rest of the brain. It was confirmed that V1 scales with positive allometry relative to LGN. A number of possible explanations for scaling are discussed. V1 scaling may have to do with the tendency of large brains to be more compartmentalized than small brains, or V1 scaling might reflect the geometry of information representation. © 2004 Wiley-Liss, Inc.

Key words: allometry; horizontal meridian; independent contrasts

Here three-dimensional reconstructions of primate primary visual cortex (V1) are used to address two topics in the evolution of primate visual systems. First, anthropoids and strepsirrhines are examined for differences in the size and shape of V1. Second, the scaling of V1 relative to the lateral geniculate nucleus (LGN), the rest of cortex, and the rest of the brain is examined.

The anthropoid visual system differs in a number of respects from that of other primates. These are generally focused on aiding in a high-acuity diurnal lifestyle and include specializations in the size and shape of the eye, orientation of the orbits, and density of cones and retinal ganglion cells (Ross, 2000; Tetreault et al., 2004).

In addition to these differences in the retina and the dioptric apparatus of the eye, several anthropoid-strepsirrhine differences have been reported in V1. Anthropoids have a larger V1 at a given body mass than strepsirrhines (Frahm et al., 1984). Anthropoids also dedicate a much larger portion of V1 to central vision (Rosa et al., 1997). The three-dimensional structure of V1 may also be different in anthropoids and nonanthropoids. One strepsirrhine, the galago, has been reported to have a V1 representation that is less elongated along the horizontal meridian than what is typical in anthropoids (Rosa et al., 1997). In this study, anthropoid-strepsirrhine differences in V1 are investigated using three-dimensional reconstructions from a number of primates. In this analysis, the effect of clade and also possible effects of activity pattern and brain size are considered, both of which can affect structure in the visual system.

A second major focus is to examine the scaling of V1 relative to LGN, the rest of cortex, and the rest of the brain. Stevens (2001) proposed that V1 neuron number in primates scales with positive allometry relative to LGN neuron number. Stevens (2001) argued that because V1 represents edge orientation explicitly, increases in x-y resolution in LGN will lead to increases in both x-y resolution and the resolution of edge orientation in V1. This would mean a disproportionate increase in the number of V1 neurons. Here, independent contrasts are used to examine

Grant sponsor: the National Institutes of Health; Grant number: EY11759; Grant sponsor: W.M. Keck Foundation for Discovery in Basic Medical Research at the California Institute of Technology.

E.C.B.'s current address is Department of Human Genetics, University of Chicago, Chicago, Illinois.

*Correspondence to: Eliot C. Bush, Department of Human Genetics, University of Chicago, 920 East 58th Street, CLSC 3rd Floor, Chicago, IL 60637. Fax: 773-834-0505.
E-mail: ebush@bsd.uchicago.edu

Received 20 May 2004; Accepted 1 July 2004
DOI 10.1002/ar.a.20114
Published online 6 October 2004 in Wiley InterScience (www.interscience.wiley.com).

the scaling of V1 and LGN volume. Also, several new measurements of LGN neuron number are presented. These are relevant to evaluating the hypothesis of Stevens (2001) because they help define how much variation there is in LGN neuron number in anthropoids.

MATERIALS AND METHODS

Reconstructions were made using histological sections from the Comparative Mammalian Brain Collection at the University of Wisconsin at Madison. These were digitized using a standard office flatbed scanner (Epson Expression 800) at 800 dpi. The resulting images were aligned to make a three-dimensional volumetric data set, with the goal of reproducing as closely as possible the original relationship between sections. To do this, pictures of the brains taken before embedding and sectioning from standard views were used. For example, to align a set of coronal slices, preembedding pictures from lateral and dorsal views were used. The profile of the brain from the lateral view provides information about where the top and bottom of each coronal section should be. A program was written to determine where each section would fall on the profile, extract information about its top and bottom position, and represent it in a guide image. One guide image for every section was produced. Each guide contained a set of lines indicating where the top, bottom, left, and right of its corresponding section should be. Each section was manually aligned to its guide image. It was also aligned with adjacent sections. This resulted in a data set that was well aligned locally and also reproduced very closely the original shape of the brain. Pre-embedding pictures of the brains were also used to calculate voxel dimensions and correct for shrinkage. Those interested in the details of this should refer to Bush and Allman (2004). The aligned data sets are available on the Web (http://allmanlab.caltech.edu/people/bush/3d-histol/3d-brain-recon.html).

One goal of the present study was to examine the shape of V1 in different primates and to relate observable differences to clade, activity pattern, or body size. To do this, a numerical measure of V1 elongation was required. From morphological reconstructions, it is possible to estimate the length of the representation of the horizontal meridian (these measurements are described in more detail below). Ideally, one would like to compare this length with a length in the direction orthogonal to the horizontal meridian. However, it is not obvious how to measure this dimension in a consistent way in different species. Instead, the surface area of V1 was measured. The ratio of the square of horizontal meridian length to surface area gave an estimate of V1 elongation. Horizontal meridian length needed to be squared in order to make it comparable to an area measure.

Surface area and length measurements were made on a subset of 14 species for which 85 or more slices per brain were available. These measurements were made using the Amira software package (TGS, San Diego CA). The aligned data set was segmented using semimanual methods. These segmentations were used to produce surface representations of the pial surface of V1. These were smoothed (using an algorithm that moves each vertex toward the average position of its neighbors) in order to remove the stair-like slice structure of the original data set, which could have led to large overestimates of surface area. The area of the smoothed surfaces was then measured. Also, the length of the horizontal meridian was estimated. In primates, the horizontal meridian follows the base of the calcarine sulcus from its rostral tip caudally. It emerges at the caudal end of the sulcus and roughly follows the trajectory defined by the sulcus all the way to the lateral edge of V1. By measuring the length of this, a crude estimate of the length of the horizontal meridian was obtained.

Volume measurements were made from a total of 22 species of primates, including 14 haplorhines and 8 strepsirrhines (see Table 1 for a listing of species). In the analysis, measurements from the present study were combined with those of Frahm et al. (1984), averaging in cases of overlap. The result is a data set of 37 primate species, including 22 haplorhines and 15 strepsirrhines. Independent contrasts were used to analyze these, and discussion of the methods for both measurement and analysis can be found in Bush and Allman (2004). Species were grouped as nocturnal, diurnal, or cathemeral (which included species that were hard to characterize) based on Rowe (1996).

Both standard regression techniques and methods involving independent contrasts were used to demonstrate grade shifts between anthropoids and strepsirrhines. Using independent contrasts, the scaling exponents for the two groups were compared by pooling the contrasts for both groups and regressing through the origin. Then, the residuals for each group were compared using a *t*-test. After establishing similarity of slope, evidence of grade differences were sought by comparing the contrast between the two groups with the other contrasts (Barton and Harvey, 2000).

The present study also contains neuron counts for LGN in human, macaque, and chimp. These measurements were done on celloidin-embedded brains, one individual per species. The macaque comes from the collection of the authors, and the human and chimp are part of the Yakovlev Collection at the Armed Forces Institute of Pathology. Neuron counts were done with the optical fractionator method on a Stereo Investigator-equipped Nikon 800 microscope.

RESULTS

V1 shape differs significantly between anthropoids and strepsirrhines. In anthropoids, V1 is a relatively elongated structure. Figure 1 is a graphic illustration showing the pial surface of V1 in an owl monkey and a ringtail lemur. The horizontal meridian goes from the lateral most edge of V1 to the rostral end of the calcarine sulcus. As can be seen in Figure 1, this distance is proportionally much longer in the owl monkey. This distance and V1 surface area were measured for 14 primates (Table 1). Figure 2A is a strip chart showing the ratio between the square of horizontal meridian length and V1 surface area. High values of this ratio correspond to greater elongation in V1 shape. Figure 2A shows data for anthropoids, strepsirrhines, all nocturnal primates, and all diurnal primates, and it reveals that anthropoids have a significantly elongated V1 relative to strepsirrhines (*t*-test $P = 0.00063$). When nocturnal and diurnal primates are compared in the same way, the two are not significantly different (*t*-test $P = 0.18$).

In Figure 2B, the same ratio is plotted against V1 gray matter volume. A cursory glance at Figure 2B may give the impression that the two variables are correlated. However, a more careful examination shows that this impression is a byproduct of the fact that the anthropoid clade

TABLE 1. Measurements in V1 and the rest of the brain for a sample of primates*

	V1G	LGN	V1surf	Hmerid	Wb	NeoW	NeoG
Hylobates lar	4.4	0.15	30.1	11.66	101.28	20.5	45.12
Pan troglodytes	11.03	0.29	65.24	18.28	364.14	103.77	163.18
Homo sapiens	16.14	0.37	NA	NA	1251.85	422.48	555.36
Cercocebus torquatus	5.12	0.2	NA	NA	117.08	40.36	47.92
Cercopithecus nictitans	4.14	0.12	NA	NA	64.74	11.81	35.99
Macaca mulatta	3.26	NA	22.18	10.02	65.76	16.35	30.54
Papio hamadryas	7.7	0.22	NA	NA	159.11	39.68	79.1
Semnopithecus entellus	4.32	0.14	28.98	9.91	107.54	24.26	51.85
Alouatta palliata	1.86	0.09	12.81	8.43	43.52	11.41	17.15
Aotus trivirgatus	0.63	NA	4.69	4.8	11.42	1.74	5.23
Ateles sp.	3.13	0.12	23.27	10.2	72.41	17.95	30.93
Callicebus sp.	0.84	0.03	6.79	5.54	11.9	1.94	5.12
Saimiri sciureus	1.43	NA	NA	NA	21.22	3.7	10.44
Tarsius syrichta	0.28	0.02	NA	NA	3.06	0.15	1.47
Eulemur mongoz	1.06	0.05	8.66	5.33	22.24	3.83	8.2
Galago senegalensis	0.22	0.01	2.64	2.82	3.58	0.23	1.34
Lemur catta	1.31	0.06	9.1	5.29	21	2.12	8.96
Microcebus murinus	0.11	0.01	1.55	2.28	1.65	0.1	0.59
Nycticebus coucang	0.58	0.03	NA	NA	11.27	1.07	4.4
Otolemur crassicaudatus	0.45	0.02	4.31	4.21	7.11	0.57	3.1
Perodicticus potto	0.33	0.02	3.16	3.25	10.16	0.64	3.91
Propithecus verreauxi	1.57	0.07	NA	NA	25.19	2.35	9.84

*V1G, V1 gray matter volume in cm^3; LGN, LGN volume in cm^3; V1surf, V1 surface area in cm^2; Hmerid, sum of distance along horizontal meridian for two hemispheres; Wb, whole brain volume in cm^3; NeoW, neocortical white matter volume in cm^3; NeoG, neocortical gray matter volume in cm^3. All measurements are for two hemispheres combined.

has larger values for both variables. Using a nonparametric correlation method (Spearman's rho), anthropoids and strepsirrhines taken separately do not show any significant relationship between these two variables ($P = 0.66$ and 0.92, respectively). In fact, even when the two groups are considered together, the correlation is not quite significant ($P = 0.06$). This shows that the relative proportions of V1 do not vary systematically with V1 size.

The volume measurements, which are given in Table 1, agree with previous conclusions that anthropoids have a larger V1 than strepsirrhines at a given body weight. Figure 2C is a plot of V1 volume vs. body mass using data of this study and that of Frahm et al. (1984) combined. Figure 2D shows V1 volume relative to LGN volume. There also appears to be a grade shift here. The slopes of the regression lines for anthropoids and strepsirrhines are not significantly different ($t = 1.31$; $P = 0.20$), but the intercepts are ($t = 2.49$; $P = 0.019$). The use of independent contrasts gives the same result. When anthropoid and strepsirrhine contrasts are regressed through the origin together, the residuals for the two groups are not significantly different ($t = 0.735$; $P = 0.47$). The slopes are therefore not significantly different. However, the absolute values of the V1 volume contrast between anthropoids and strepsirrhines are larger than all the other contrasts (1.9 standard deviations greater than the mean). For a given LGN volume, anthropoids tend to have a larger V1 than strepsirrhines.

Independent contrast analysis shows that V1 scales with negative allometry relative to the rest of cortex and the subcortical brain. V1 gray matter contrasts scale with the rest of cortical gray matter contrasts to the 0.74 power (95% CI = 0.62–0.85). V1 scales with the subcortical brain to the 0.80 power (95% CI = 0.69–0.91). Independent contrasts were also used to examine the scaling of V1 to LGN. It was found that V1 scales with LGN to the 1.11 power (95% CI = 1.02–1.21).

Finally, neuron counts for LGN were provided in three species of primates (Table 2). LGN neuron numbers were similar in human, chimp, and macaque.

DISCUSSION

The results of the present study showed that the three-dimensional structure of V1 is significantly different in anthropoids and strepsirrhines. V1 in anthropoids is more elongated than in strepsirrhines. This difference is not a byproduct of differences in absolute V1 size or of differences in activity pattern. There is no systematic relationship between absolute V1 size and elongation, and nocturnal and diurnal primates do not differ significantly in V1 elongation.

What could cause this clade-specific difference in V1 shape? One possibility is that it reflects a combination of developmental constraints and changes in the structure of anthropoid extrastriate cortex. At its rostral end, V1 borders on a part of limbic cortex that has been called area prostriata (Allman and Kaas, 1971; Sanides, 1972). This border is found in many mammals, but its presence in primates is interesting. Primates have a large number of extrastriate visual areas, and one wonders if it would not be more economical for V1 to border entirely on those areas. Perhaps something about the development of cortex constrains the rostral end of V1 to border on area prostriata, with that area acting as a sort of anchor. If this were true, rearrangement in extrastriate visual areas could have forced V1 to become more elongated in anthropoids.

Another interpretation is based on the fact that the strip-like shape of anthropoid V1 reduces the distance from a point in the center of the area to its border. This could mean that the distance between visuotopically equivalent points in V1 and V2 is reduced when V1 is more elongated. Anthropoids probably have more cortical visual areas than strepsirrhines. Perhaps as anthropoids

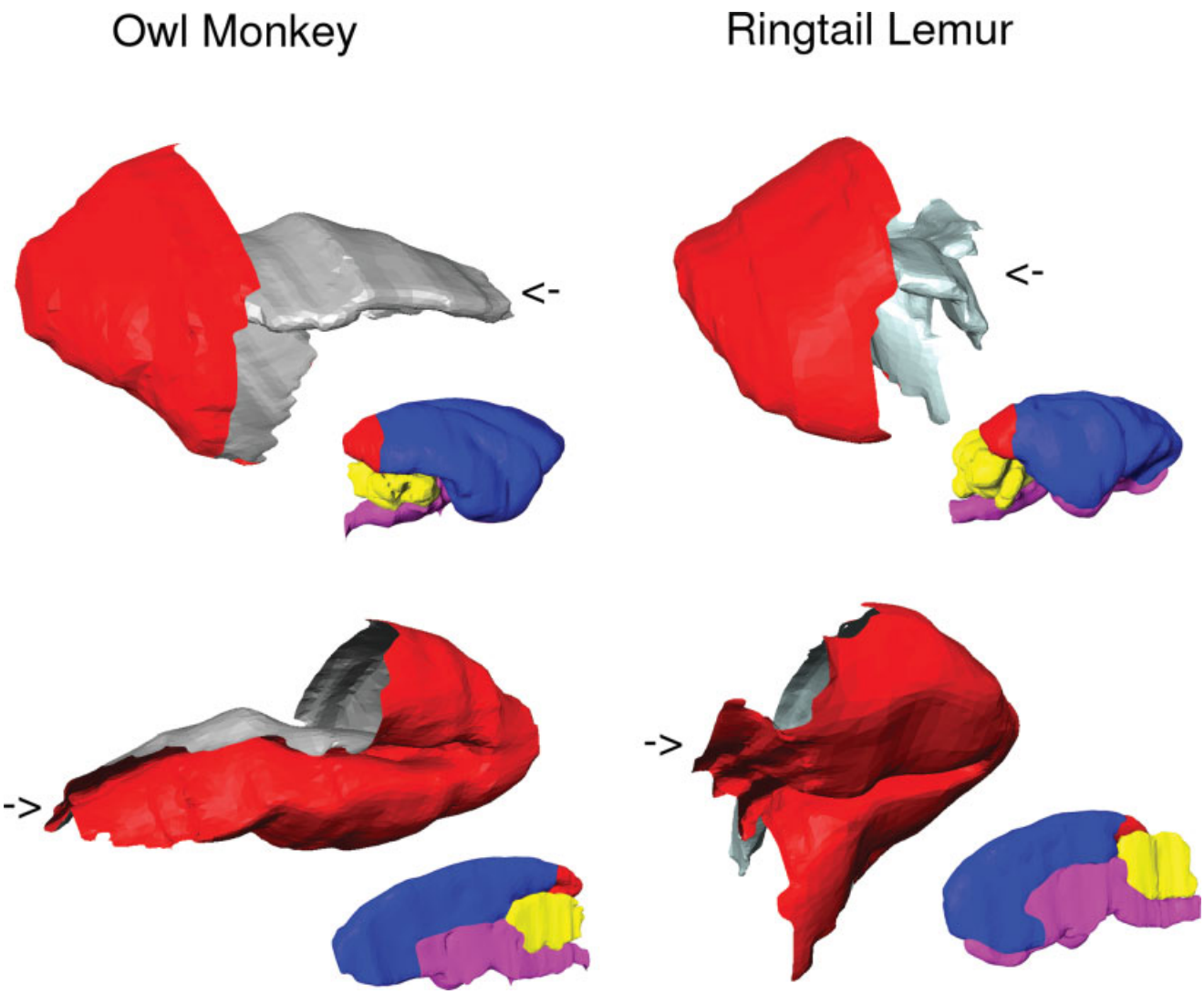

Fig. 1. An illustration of the pial surface of V1 in two primates, the owl monkey (an anthropoid) and the ringtail lemur (a strepsirrhine). The outer side of this surface is colored red, and the inner side gray. At the lower right of each V1 image is a small image of one hemisphere showing what orientation V1 is being viewed from.

acquired more areas, there was pressure to cut down the communication time between V1 and V2.

Figure 2C confirms that anthropoids have a larger V1 than strepsirrhines at a given body size (Frahm et al., 1984). Here a similar comparison with LGN volume was made. This new comparison is interesting because LGN is the principal source of V1's input. Results also show that for a given LGN size, anthropoids have a larger V1 than strepsirrhines. The difference represents a grade shift between the two clades. As one can see from Figure 2D, differences in activity pattern do not seem to account for it. The increase in anthropoid V1 relative to LGN suggests the addition of new processing machinery in anthropoids. Anthropoid V1 may simply be doing more with the same input.

The second major focus of this study related to the scaling of V1. The scaling between V1 and LGN volume confirmed with independent contrasts that V1 scales with positive allometry relative to LGN. This is broadly consistent with hypothesis of Stevens (2001). Also, LGN neuron counts suggest that humans, chimps, and macaques have roughly the same number of LGN neurons. This similarity is consistent with what is known about retinal ganglion cell number in diurnal anthropoids (Table 3). The general suggestion is that diurnal anthropoids all have roughly the same number of retinal ganglion cells and LGN neurons. If this is true, it makes the hypothesis of Stevens (2001) more difficult to test. If most primate variation in LGN and RGC number exists between anthropoids and strepsirrhines, it will be difficult to show that disproportionate increases in V1 neuron number result from of a systematic scaling relationship rather than two independent grade shifts. Independent grade shifts might have caused anthropoids to have more LGN neurons and dis-

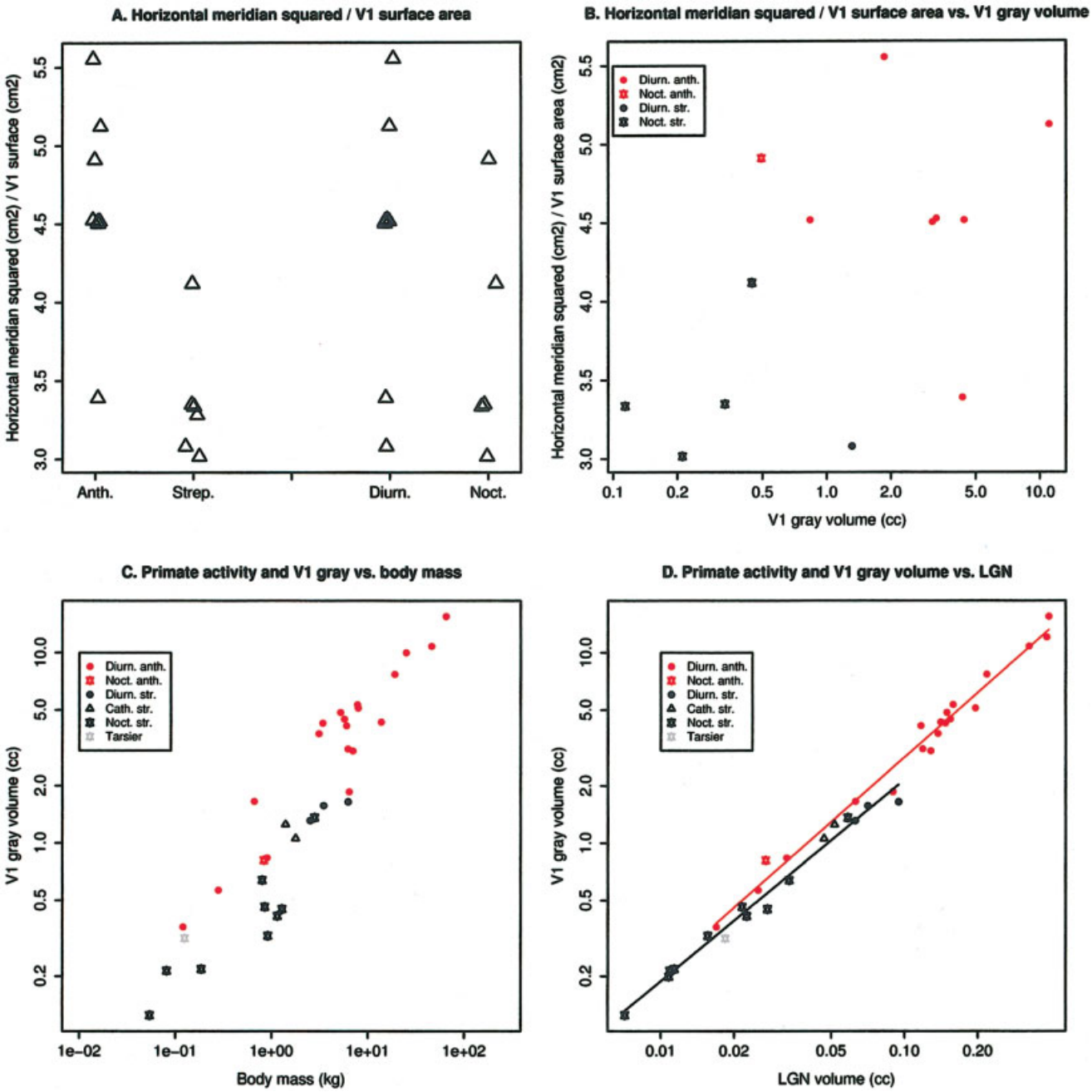

Fig. 2. **A:** Strip chart showing the ratio of horizontal meridian squared to V1 surface area. Anthropoids and strepsirrhines are shown, as well as all diurnal and nocturnal primates. Symbols have been jittered slightly so that overlapping points can be distinguished. **B:** The same ratio as in A, plotted against V1 volume. For **C** and **D**, V1 and LGN volume data are from data of this study combined with that of Frahm et al. (1984). Body mass comes from Stephan et al. (1981) and Silva and Downing (1995). C: V1 volume vs. body mass for anthropoids and strepsirrhines. D: V1 volume vs. LGN volume. Least squares regression lines are included.

proportionately more V1 neurons. If LGN neuron number variation exists mostly between anthropoids and strepsirrhines, it will be hard to distinguish between the possibility of two grade shifts and the more systematic relationship described by Stevens (2001).

Let us briefly return to the grade shift in V1 volume between anthropoids and strepsirrhines. Anthropoids tend to have larger LGNs than strepsirrhines. It is possible that part of the grade difference reported in this study is due to the factors proposed by Stevens (2001). However, note that in Figure 2D, a number of anthropoids overlap with strepsirrhines in terms of LGN size. In these cases, the anthropoids still have a larger V1.

TABLE 2. Neuron counts and coefficients of error for human, chimp, and macaque LGN

	Magnocellular		Parvocellular	
	Count	Coefficient of error	Count	Coefficient of error
Homo sapiens	127,000	0.05	1,025,000	0.06
Pan troglodytes	145,000	0.04	1,340,000	0.05
Macaca mulatta	163,000	0.05	1,302,000	0.06

TABLE 3. Primate retinal ganglion cell counts taken from the literature

	Count	Reference
Microcebus murinus	565,000	Dkhissi-Benyahya et al. (2001)
Cheirogaleus medius	192,000	Tetreault et al. (2004)
Galago senegalensis	519,000	DeBruyn et al. (1980)
Otolemur crassicaudatus	755,000	DeBruyn et al. (1980)
Tarsius syrichta	797,000	Tetreault et al. (2004)
Aotus trivirgatus	465,000	Webb and Kass (1976); Silveira et al. (1993); Jones (1965); Jacobs (1977)
Cebus apella	1,370,000	Silveira et al. (1989); Perry and Cowey (1985)
Macaca mulatta	1,600,000	Perry and Cowey (1985)
Macaca fascicularis	1,583,000	Wässle et al. (1990); Reese and Ho (1988)
Papio anubis	1,580,000	Fischer and Kirby (1991)
Cercopithecus aethiops	1,229,000	Herbin et al. (1997)
Homo sapiens	1,275,000	Van Buren (1963); Jonas et al. (1992); Curcio et al. (1990)

Systematic relationships with brain size are a prominent feature of variation in mammalian brains. The results of independent contrasts confirm that V1 scales with negative allometry relative to the rest of cortex and the rest of the brain. How are we to understand this?

One popular idea is that allometry in the brain results from developmental constraints. It is hypothesized that a fixed order of neurogenesis means that as brains get larger, those regions where neurogenesis stops first will tend to scale with negative allometry relative to the rest of the brain (Finlay and Darlington, 1995). In the case of V1, this hypothesis is not consistent with the data. In primates, V1 develops later than a number of other cortical structures (Rakic, 1988). According to the fixed order of neurogenesis theory, this should lead to V1 hyperscaling.

Another possibility is that V1 scaling has to do with the tendency of large brains to have more areas than small brains. The idea is that as an area gets larger, it becomes more costly and time-consuming for its disparate parts to communicate with each other. As a result, there may be strong pressure for certain functions to be divided, resulting in multiple areas (Kaas, 2000). In small mammals, V1 is involved in many visual functions, including global comparisons between different regions of the visual field. As V1 gets larger, it could be more efficient to move some of these global functions to new, smaller areas specialized for them, thereby economizing on wire. There is much to recommend this explanation. However, it seems to us that it cannot be the only factor explaining the scaling of V1 to the rest of cortex. One manifestation of the tendency of V1 to scale down relative to the rest of cortex can be seen in the ratio of V1 volume to total cortex volume in humans and macaques. Human V1 is a smaller proportion of total cortex than macaque V1. To explain this using the above hypothesis, one must conclude that human V1 is performing significantly less functions than macaque V1. This appears unlikely. There have been reports of histological differences in V1 between monkeys and humans (Preuss et al., 1999). But these differences do not suggest that human V1 is doing significantly less work. There must be some other explanation that accounts for the human macaque difference in V1 over total cortex volume.

Another possibility is that scaling might reflect the geometry of information representation. The most specific version of this theory is the V1-LGN hypothesis discussed above. But information representation has the potential to be a general explanation for scaling in the brain. One could imagine that in the same way that the information represented by V1 causes it to scale up relative to LGN, extrastriate areas might scale up relative to V1. To pursue this further will require thought about the nature of information processing in areas such as V2 and V4 compared with V1.

This study discussed several aspects of V1 evolution in primates and added to a growing body of evidence for unique anthropoid adaptations in V1. Also, it was confirmed that V1 scales with positive allometry relative to LGN, and negative allometry relative to most of the brain. A better knowledge of information processing in V1 would contribute enormously to an understanding of both of these topics. Fortunately, substantial resources continue to be put into studying V1. In the future, it should be possible to understand anthropoid-strepsirrhine differences on a very detailed level. Relating this understanding to differences in behavior and ecology between the two groups is an exciting prospect. Similarly, better knowledge of V1's workings should greatly clarify the puzzle of scaling.

ACKNOWLEDGMENTS

The authors thank Wally Welker, Inge Siggelkow, and Archie Fobbs for access to the Wisconsin brain collection and Kebreten Manaye for use of her Stereo Investigator.

LITERATURE CITED

Allman JM, Kaas JH. 1971. Representation of the visual field in striate and adjoining cortex of the owl monkey *Aotus trivirgatus*. Brain Res 35:89–106.

Barton R, Harvey P. 2000. Mosaic evolution of brain structures in mammals. Nature 405:1055–1058.

Bush EC, Allman JM. 2004. The scaling of frontal cortex in primates and carnivores. Proc Nat Acad Sci USA 101:3962–3966.

Curcio CA, Sloan KR, Kalina RE, Hendrickson AE. 1990. Human photoreceptor topography. J Comp Neurol 292:497–523.

DeBruyn EJ, Wise VL, Casagrande VA. 1980. The size and topographic arrangement of retinal ganglion cells in the galago. Vision Res 20:313–327.

Dkissi-Benyahya O, Szel A, Degrip WJ, Cooper HM. 2001. Short and mid-wavelength cone distribution in a nocturnal strepsirrhine primate (*Microcebus murinus*). J Comp Neurol 438:490–504.

Finlay BL, Darlington RB. 1995. Linked regularities in the development and evolution of mammalian brains. Science 268:1578–1584.

Fischer QS, Kirby MA. 1991. Number and distribution of retinal ganglion cells in Anubis baboons (*Papio anubis*). Brain Behav Evol 37:189–203.

Frahm HD, Stephan H, Baron G. 1984. Comparison of brain structure volumes in insectivora and primates: V, area striata (AS). J Hirnforsch 25:537–557.

Herbin M, Boire D, Ptito M. 1997. Size and distribution of retinal ganglion cells in the St. Kitts green monkey (*Cercopithecus aethiops sabeus*). J Comp Neurol 383:459–472.

Jacobs GH. 1977. Visual capacities of the owl monkey (*Aotus trivirgatus*): II, spatial contrast sensitivity. Vision Res 17:821–825.

Jonas JB, Schmidt AM, Müller-Bergh JA, Schlötzer-Schrehardt UM, Naumann GOH. 1992. Human optic nerve fiber count and optic disc size. Invest Ophthalmol Vis Sci 33:2012–2018.

Jones AE. 1965. The retinal structure of (*Aotus trivirgatus*) the owl monkey. J Comp Neurol 125:19–28.

Kaas JH. 2000. Why is brain size so important: design problems and solutions as neocortex gets bigger or smaller. Brain Mind 1:7–23.

Perry VH, Cowey A. 1985. The ganglion cell and cone distributions in the monkey's retina: implications for central magnification factors. Vision Res 25:1795–1810.

Preuss TM, Qi H, Kaas JH. 1999. Distinctive compartmental organization of human primary visual cortex. Proc Nat Acad Sci USA 96:11601–11606.

Rakic P. 1988. Specification of cerebral cortical areas. Science 241: 170–176.

Reese BE, Ho KY. 1988. Axon diameter distributions across the monkey's optic nerve. Neuroscience 27:205–214.

Rosa M, Casagrande V, Preuss TM, Kaas JH. 1997. Visual field representation in striate and prestriate cortices of a prosimian primate (*Galago garnetti*). J Neurophysiol 77:3193–3217.

Ross CF. 2000. Into the light: the origin of Anthropoidea. Ann Rev Anthropol 29:147–194.

Rowe N. 1996. The pictorial guide to the living primates. East Hampton: Pogonias Press.

Sanides F. 1972. Representation in the cerebral cortex and its areal lamination patterns. In: Boune G, editor. Structure and function of the nervous system. New York: Academic Press. p 329–453.

Silva M, Downing JA. 1995. CRC handbook of mammalian body masses. Boca Raton, FL: CRC Press.

Silveira LCL, Picanço-Diniz CW, Sampaio LFS, Oswaldo-Cruz E. 1989. Retinal ganglion cell distribution in the Cebus monkey: a comparison with the cortical magnification factors. Vision Res 29: 1471–1483.

Silveira LCL, Perry VH, Yamada ES. 1993. The retinal ganglion cell distribution and the representation of the visual field in area 17 of the owl monkey, *Aotus trivirgatus*. Visual Neurosci 10:887–897.

Stephan H, Frahm H, Baron G. 1981. New and revised data on volumes of brain structures in insectivores and primates. Folia Primatol 35:1–29.

Stevens C. 2001. An evolutionary scaling law for the primate visual system and its basis in cortical function. Nature 411:193–195.

Tetreault N, Hakeem A, Allman JM. 2004. The distribution and size of retinal ganglion cells in *Cheirogaleus medius and Tarsius syrichta*: implications for the evolution of sensory systems in primates. In: Ross C, Kay R, editors. Anthropoid origins: new visions. New York: Kluwer/Plenum. p 463–475.

Wässle H, Grünert U, Röhrenbeck J, Boycott BB. 1990. Retinal ganglion cell density and cortical magnification factor in the primate. Vision Res 11:1897–1911.

Webb SV, Kaas JH. 1976. The sizes and distribution of ganglion cells in the retina of the owl monkey, *Aotus trivirgatus*. Vision Res 16:1247–1254.

Van Buren JM. 1963. The retinal ganglion cell layer. Springfield, IL: Charles C. Thomas.